VOLUME LV JULY, 1945 NUMBER 7

THE LARYNGOSCOPE

FOUNDED IN 1896
BY
MAX A. GOLDSTEIN, M. D.

PUBLISHED BY
THE LARYNGOSCOPE
640 SOUTH KINGSHIGHWAY ST. LOUIS (10), MO., U. S. A.

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THE
LARYNGOSCOPE.

VOL. LV JULY, 1945. No. 7

THE THERAPY OF HISTAMINE AND NICOTINIC ACID.

JOHN J. SHEA, M.D.,
Memphis, Tenn.

The therapy of histamine and nicotinic acid has rendered marvelous relief to many patients suffering from the dysfunctions of the auditory nerve, manifested by deafness, tinnitus or vertigo, and to headache of the nonsurgical variety. These drugs are vasodilators and as such relieve vasoconstriction affecting the auditory nerve and cranial vessels. Nicotinamide does not produce the cerebral vasodilatation like nicotinic acid, and the *modus operandi* of nicotinic acid accomplishing vasodilatation is a result of the chemical combustion when it becomes an amide.

A foreign substance capable of sensitizing body cells is called an allergen (antigen). Allergens may enter the body by absorption through any of the mucous membranes or through the skin; through or into the skin by injection; or by absorption from some focus of infection. Although it is not known why some persons become allergic to certain substances and others do not, the causes of allergic reactions are fairly well understood. When an allergen (antigen) enters the body of certain persons for the first time, it comes in contact with cells of the reticuloendothelial system. It then becomes oriented with part of the cell globulin, forming antibodies specific for the allergen. These antibodies are probably developed by the allergic person in an attempt to neutralize the allergen and are believed to become anchored to the tissue cells themselves. When this occurs, the patient

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 18, 1945.

is said to be sensitized and will react to contact with the allergens which originally stimulated formation of the antibodies. When a second exposure to the same allergen (antigen) occurs, the allergen causes a violent disturbance at the surface of the cell. As a result of the injury to the cell membrane (histamine H-substances), a normal constituent of body cells, is released from the site of its intracellular fixation and is diffused into the surrounding tissue fluid and general circulation. This free histamine causes reactions of different types in various parts of the body. In some allergic persons the nasal mucosa is affected, producing symptoms of rhinitis or hay fever. In others, histamine may reach the mucosa of the lungs causing asthma, or the skin causing hives, or angioneurotic edema. In short, the histamine theory of allergic reactions simply means that, in allergic conditions usually seen by the clinician and caused by nonliving protein antigens (regardless of which ones), the factor responsible for allergic symptoms is histamine (H-substance).

HISTAMINE.

Barger and Dale, in 1910, at the Wellcome Physiological Research Laboratories isolated and synthesized histamine while investigating the active principles of ergot. Further studies of its pharmacological and clinical properties resulted in its use: *a.* in the treatment of rheumatism; *b.* as a stimulant for gastric secretions while testing stomach contents; *c.* as a nonspecific desensitizer in allergic conditions; *d.* for the relief of vascular headaches; *i.e.*, in the treatment of Ménière's syndrome.

Histamine will stimulate any of the body tissues and increases their secretions, including the cerebral spinal fluid. Administered orally, the gastric fluid renders it inert, being destroyed by an enzyme system, histaminase, which is secreted by the intestinal mucosa. Injected subcutaneously or intravenously, it produces marked stimulation of the tone and rhythm of smooth muscle similar to that of pituitary extract but with a fall of the blood pressure. The increase of cerebrospinal fluid is the result of dilatations of the vessels of the meninges and the brain, and the disturbed intracranial blood pressure is the cause of the accompanying headache. Histamine is rapidly absorbed, but it is active only when

administered parenterally and is rapidly destroyed, with a small quantity being excreted in the urine. Its toxic symptoms are a very rapid drop in the blood pressure, vasodilatation of the vessels in the skin, intense headache, visual disturbances, bronchial constriction and dyspnea and, in severe cases, shock.

Epinephrine is its specific antagonist and should always be available when the drug is administered.

It is not the purpose of this paper to consider the rôle of histamine in the treatment of rheumatism or as a stimulant of gastric secretions, but rather to review its application for the relief of histamine cephalgia, Ménière's syndrome and as a common denominator in the desensitization of allergic conditions.

Horton and his co-workers at the Mayo Clinic have successfully developed histamine therapy as applied to our specialty. Atkinson added the principles of testing, while others, like Moore, Farmer and Kaufman, have added to the clinical application of the therapy. It has been our personal experience to have treated over three hundred patients with histamine and nicotinic acid therapy. These cases are purposely not being put into statistic form because the conclusions cannot be crystallized into transparent facts. As yet there is no clinical finding or laboratory test which will accurately choose between histamine and nicotinic acid therapy.

INTRINSIC THERAPY.

Williams, quoting Lewis, contends that physical irritants such as ultraviolet will produce a reaction similar to histamine and, when in the muscle, is known as myalgia. Sensitivity to heat, cold, change in atmospheric pressure, endocrine dysfunction and emotional disturbances are examples of intrinsic or physical allergy. The association of infection and pain in the head does not exclude allergy. Some of the worst cases of allergy develop in patients who were formerly sufferers from infection of their sinuses or mastoids, and even though surgery has eradicated the pathology, the headache or otalgia remains, because it is due to the allergy.

HEADACHE.

Headache and neuralgia are often confused. The latter is the result of pain along the cranial nerves, and the former about the distribution of the intracranial blood vessels. Ten per cent of headaches are ocular in origin (Gradle), while only three per cent are nasal (Spriggs).

Boies classifies headaches as: *1.* independent of disease of an individual organ or any general disease; *2.* headaches associated with individual organs, brain, eyes, sinuses, gastro-intestinal, kidney, pelvic organs, etc.; and a third group, those associated with general diseases. Headache is usually the result of a dilatation of the blood vessels of the dura and pia or of the venous sinuses of the dura. Cranial neuralgias follow the distribution of the Vth, VIIth, IXth and Xth cranal nerves. Of these, the Vth supplies the greatest distribution — the nasal branch of the ophthalmic to the dura in the anterior fossa; a meningeal branch from the ophthalmic to the tentorium; a branch from the second division accompanies the middle meningeal artery, while a meningeal branch of the mandibular and the spinous may be distributed to the middle fossa. The dura of the posterior fossa is supplied by a meningeal branch from the vagus and hypoglossal.

Headache is not the result of an increase of cerebral spinal fluid pressure unless it is accompanied by a dilatation of the cerebral blood vessels, because the headache is the result of stretching of the pain end-organs of the cranial blood vessels, or the stretching of the pain end-organs of the ventricular subarachnoid space.

HISTAMINIC CEPHALGIA.

Horton under this term recognized a definite clinical picture consisting of a unilateral headache with sudden onset, involving the temple, eye and, at times, the face and neck. The attacks are usually sharp and severe. The signs and symptoms are limited to the distribution of the carotid arteries. The phenomenon can be duplicated by the administration of histamine and relieved by epinephrine. The absence of visual disturbances, nausea and vomiting differentiates the attacks from migraine and seldom is there an inherited tendency. The absence of trigger areas and the fact that the distribution is vascular and not confined to the branches of

the trifacial nerve distinguish it from tic douloureux. The attacks are more frequent when the patient is lying down, and the habit of sleeping flat encourages the headache. Some patients are relieved by sitting up, while others must arise and walk. The treatment suggested by Horton is as follows: ampoules containing 0.1 mgm. of histamine base (0.276 mgm. of histamine acid phosphate) in 1 cc. are used, and injections are given subcutaneously (not intravenously or intracutaneously) twice daily, over a period of from 10 days to three weeks.

First day	First injection	0.25 cc.
	Second injection	0.30 cc.
Second day	Third injection	0.35 cc.
	Fourth injection	0.40 cc.
Third day	Fifth injection	0.45 cc.
	Sixth injection	0 50 cc.
Fourth day	Seventh injection	0.55 cc.
	Eighth injection	0.60 cc.
Fifth day	Ninth injection	0.65 cc.
	Tenth injection	0.70 cc.
Sixth day	Eleventh injection	0.75 cc.
	Twelfth injection	0.80 cc.
Seventh day	Thirteenth injection	0.85 cc.
	Fourteenth injection	0 90 cc.
Eighth day	Fifteenth injection	0.95 cc.
	Sixteenth injection	1.00 cc.
Ninth day	Seventeenth injection	1.00 cc.
	Eighteenth injection	1.00 cc.
Tenth day	Nineteenth injection	1.00 cc.
	Twentieth injection	1.00 cc.

Overdosage must be avoided, and if a slight flushing or other symptoms indicating histamine effect occurs, the next dose should be reduced 50 per cent, and an attempt made gradually to increase the dose.

When relief from attacks have been achieved, a maintenance dose to prevent recurrence should be established by giving the patient about 1 cc., one to three times weekly. Average maintenance dose is two injections weekly, but some few patients will be found to get along without it.

Case 1: Mrs. H., aged 28. Chief complaint, awakened at night by a headache, which demands that for relief she assume a sitting posture or, better, to get out of bed and walk about the room. The pain would be confined to the right side and equally distributed behind the eye and down into the neck. Previous attacks of sinusitis denied and there was no evidence of clinical sinus disease. Histamine test was positive. Six weeks of histamine therapy relieved the symptoms.

Case 2: Mr. A. B. T., aged 32, was first seen with a chronic maxillary sinusitis of dental origin, which was cared for surgically; but the patient continued to complain of headaches that awakened him at night and

though relieved by getting up, would return upon lying down. The antrum had healed normally and no sign of infection remained. A histamine test was positive, and after two weeks of therapy, the headaches were greatly relieved, and at the end of a month were absent.

MENIERE'S SYNDROME.

Ménière, in 1861, presented a disease entity which he thought was the result of a hemorrhage into the internal ear. Though pathologically incorrect, he established a clinical triad, recurring vertigo, deafness and tinnitus, which remains as the characteristics of the disease.

The treatment of this syndrome has included purgation by hepatic stimulants, salt-free diet, reduction of fluid intake, displacement of the sodium by the K. ion and histamine therapy.

Procedure: A complete history is taken to determine the type of vertigo and its relationship to the tinnitus and deafness. A thorough examination is made of the ears, nose and throat including catheterization of the Eustachian tubes. The hearing tests include the tuning forks with special reference to the Renne and Schwabach tests, and an audiometric study by bone and air conduction. The study of the vestibular apparatus is restricted to the caloric test, using ice water. The patient is placed in the supine position with the head rotated to first the left, to study the right ear and after an interval of rest turned towards the right to study the left ear. Following the method of Atkinson, an intradermal test is made with 0.1 cc. of 1:20,000 dilution of histamine base, and to be considered sensitive to histamine the reaction should exceed half an inch with pseudopod and remain for 15 minutes. One-half cc. of histamine acid phospate, 2.75 mgm. in 5 cc., is given subcutaneously to observe the degree of headache or vertigo.

If a headache is produced and is histaminic, it should be cured by the administration of 3 to 5 minims of epinephrine.

Case 3: Mr. R. L. B., aged 47, sought relief from vertigo and is an example of the male hypoendocrine individual. From October, 1943, to January, 1944, his vertigo was partially controlled by hypodermatical administration of histamine. In January, 1944, a series of three intravenous injections gave temporary improvement. Not being satisfied with his progress, nicamin therapy was carried out for three months, then a rest from all medicine for four months, when hapamine was used with moderate improvement. Comparing the nicotinic acid to the histamine,

we decided in favor of the nicotinic acid supplemented by the addition or oreton twice a week. This individual has improved physically and mentally. His present medication consists of nicotinic acid 100 mgm. orally whenever he feels its need. At times he can abstain from all medicine for a week but at times he requires four or five doses of the nicotinic acid.

TREATMENT.

Horton considers the treatment as primary, "eradicating the acute symptoms of vertigo, nausea, vomiting and tinnitus by giving intravenously 1 mgm. of histamine base in 250 cc. of physiological saline solution . . . at the rate of 50 or 60 drops per minute by the gravity method." These injections may be repeated daily for three doses and "the second phase of the problem resolves itself into the prevention of future attacks . . . by adequate maintenance dose."

The amount and length of time necessary for desensitization is variable. It is our practice to try 0.5 cc. of a solution which contains 2.75 mgm. in 5 cc. of histamine acid phosphate twice a week. Some patients cannot tolerate this amount and others require more. Recently, a prominent surgeon told me it required 3 cc. daily for two weeks to eliminate his vertigo. As an adjunct treatment an initial dose of calomel gr. 3 and sodium bicarbonate gr. 10, followed by a saline purge, will prepare the bilious patient for the histamine therapy. During the period of administration, histaminase (torantil 10 units) one tablet ofter two meals a day assists in the destruction of the histamine already within the intestine. To eliminate the influence of the hepatic toxins, it is advisable to give each night the following prescription: tr. belladonna 10, tr. cardamon comp. 10, sodium bromide 10, cascara evac. 10, diazyme 30, chologestin qs ad 180. Sig: Two teaspoonfuls in water each night.

It is better to prolong the treatment than to cut it short and to advise the patient of the benefits of early repetition of dosage rather than procrastinating. Following the suggestion of Moore, if the Eustachian tubes are found to be partially closed they should be catheterized and dilated with a bougie.

Case 4: Mr. E. C. R., aged 42. Chief complaint, vertigo and staggering with increasing deafness and tinnitus in the right ear. General examination was negative, although gall bladder disease was suspected. Caloric test: after one minute, the right developed a slow rotary nystagmus which changed to a horizontal on change of position, while the left was

active in 30 seconds. All spinal fluid tests were negative. There was a marked improvement after three intravenous injections of 1 mgm. of histamine base. The maintenance dose had to be increased to 1 cc. of the 5 cc. (equals 2.75 mgm.) of histamine acid phosphate. This man can now enjoy horseback riding and is leading a normal life.

HEPATIC INFLUENCES.

During the treatment of histamine-sensitive patients, one will encounter individuals who have a hepatic disease requiring surgery, and until this surgery is successful, the histamine treatment can be only temporary and inadequate, such as the following case.

Case 5: Miss M. F., aged 46, was admitted with active vertigo and a severe intracranial pain. Her history was that of being allergic to many foods, especially to wheat. Examination of the ears revealed a moderate perceptive deafness and her histamine test was positive. This patient was greatly relieved by a series of three doses of intravenous histamine but could not be maintained by subcutaneous doses. She developed an acute gall bladder attack, which required surgery. Following the removal of the gall bladder, she was controlled by maintenance doses of 0.5 cc. of histamine acid phosphate (2.75 mgm. in 5 cc.) twice a week.

HISTAMINE IN THE TREATMENT OF ALLERGY.

Relief from allergy should be obtained by abstaining from foods or avoiding the environments containing allergens to which the patient is sensitive. Such abstinence is often difficult, and the avoidance impossible. Desensitization against the contact allergens such as dust, powder and fungi is expensive and tiring, but necessary.

Farmer and Kaufman, quoting Dale's theory of anaphylaxis, emphasize the importance of the liberation of histamine during an attack of allergy. If a tolerance to histamine can be produced in an allergic individual, the attacks will be lessened. Following this line of thought, they suggested a therapy of minute doses starting with 1 to 1,000 mgm., increasing 50 per cent if tolerated and administered two or three times a week. As the dose increases the intervals between injections increases, the sustaining doses being given once a week to once a month in an effort to achieve the best results with the smallest amount of histamine.

Case 6: Mrs. V. S. L., aged 49, had as her chief complaint recurrent attacks of sinusitis and tinnitus. She had been studied in several of the larger clinics without results. Examination: nasal membranes pale, swollen and turbinates uniformly enlarged. Roentgenological study of her sinuses revealed an arrested type of frontal sinus and a uniform cloudiness +2 of the antra. The sphenoids were negative and irrigation

of the antra through the natural ostii produced a small amount of clear mucus with an eosinophil count of 16 cells. Allergic sensitization tests isolated a moderate number of foods to which she was sensitive (onion, Irish potato, orange, chocolate, shell fish, berries, mushrooms, coconut, cotton seed and tobacco). Zinc ionization of the membranes of the nasal cavity and antra was successful and eliminated the symptoms of sneezing and profuse nasal discharge, but the headache persisted. The histamine test was negative. The initial dose increased her headache but after a month's desensitization she was free of the headache and tinnitus.

NICOTINIC ACID.

In searching for a substitute for histamine, Harris and Moore suggested the use of nicotinic acid. A patient under the influence of nicotinic acid resembles one who has received a full therapeutic dose of aconite. If the vasodilatation is manifested in the splanchic system, there will be abdominal distress and at times a fainting sensation. Deficiency of nicotinic acid results in profound alteration of body chemistry and in tissue changes which involve the nervous system and digestive tract. Knowledge of the distribution of nicamin in foods is limited — meats are the most important source, with liver and pork leading the list. It is difficult to obtain sufficient quantities of nicotinic acid unless meat is included in the diet. The initial dose given hypodermatically is 25 mgm., with the patient lying down. Subsequent administration may be a second 25 mgm. dose with an interval of 20 to 30 minutes between doses to allow a readjustment of the circulation. As a rule the hypodermatical injections may be on alternate days, spaced by oral doses of 50 to 100 mgm. Like histamine, there is no definite indication for the choice of nicotinic acid and each patient must be observed for signs of improvement. Many of the seasonal allergic individuals will tolerate 50 mgm. daily, hypodermatically, with relief of their sneezing and nasal blockage. Those suffering from the spring pollens react better than those from the fall pollens.

Nicotinamide does not produce the same vasodilatation and is of no value in the treatment of vertigo or the congested types of headaches. It is of value when an intravenous injection is needed for pellagra patients, and in the treatment of Vincent's angina or other ulcerated lesions of the throat.

Case 7: Mrs. C. S., aged 45, a chronic sufferer from the pollens of the spring trees and grasses. Attempts at desensitization were never successful. At the onset of the symptoms in the spring of 1943, nicamin 2 cc. (equals 50 mgm.) produced a terrific reaction manifested by an extensive flushing of the skin and a smothering sensation. The dose

was repeated daily for 36 days with satisfactory relief of her allergy. During the month of September, 25 doses were administered with less relief from her fall allergy. The oral administration could not be tolerated because of the gastric distress.

Case 8: Mrs. R. C. G. was a duplicate in Case 7, in that she received greater relief in the spring than in the fall, and was able to tolerate the oral tablets.

HAPAMINE.

Fell and his associates believed that if histamine could be chemically combined with some antigenic substance, a compound would be formed which would stimulate formation of antibodies specific for histamine. After extensive chemical experimentation, these workers found that histamine could be combined with a protein to form an antigenic complex. Further laboratory experiments demonstrated that when administered parenterally, this combination of histamine and globulin, known as hapamine, is capable of stimulating formation of histamine antibodies without producing typical histamine reactions.

When hapamine is administered to the allergic patient, it combines with certain cellular substances forming antibodies which are capable of neutralizing histamine. By the injection of increasing amounts of hapamine, the patient's body cells are stimulated to produce antibodies which confer a certain degree of immunity to histamine — an immunity similar to that developed by a child when immunized against diphtheria or pertussis. These hapamine (histamine) antibodies remain inactive until the allergic patient comes in contact with an allergen to which he is susceptible. When this occurs, histamine is released, just as in "nonimmune" patients; but typical allergic symptoms do not develop since the histamine is immediately neutralized by the hapamine antibodies.

Cohen and Friedman recently investigated the antibody response to hapamine by human subjects and found that when allergic patients were given hapamine parenterally, antibodies formed which at least were partially specific for histamine. In the majority of patients, hapamine produced a capacity for very rapid histamine neutralization.

Sheldon, Fell, Johnston and Howe treated a group of patients representing cases of atopic eczema and contact dermatitis, physical allergy, seasonal hay fever, perennial aller-

gic rhinitis, gastrointestinal allergy and migrainous headache. The authors found that hapamine was of particular value in the treatment of contact dermatitis and was more promising than any other form of therapy known to them. In the opinion of Cohen, urticaria and angioneurotic edema are true allergic conditions mediated by an antigen-antibody reaction. This author feels that hapamine is of particular value in the treatment of such conditions, especially when the etiology is unknown.

Dosage: The initial dose of hapamine should be 0.01 to 0.02 cc., intradermally. If little or no reaction occurs, 0.05 to 0.1 cc. can be given subcutaneously and similar treatments given every four or five days. The dosage can be increased by 0.05 to 0.1 cc. at each injection until 1 to 1.5 cc. is being given at each dose. If intolerance is noted, dosage should be materially reduced, or treatment withheld for two weeks, after which treatment is resumed with a small dose (0.05 to 0.1 cc.) and the schedule of increase followed as recommended above. When improvement is evident the interval between doses may be increased to one week or longer. If symptoms return, regular treatment should be resumed, preferably by starting the dosage schedule in the lowest range suggested above.

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FALL REFRESHER COURSE IN LARYNGOLOGY, RHINOLOGY AND OTOLOGY AT UNIVERSITY OF ILLINOIS COLLEGE OF MEDICINE.

The University of Illinois College of Medicine announces its sixth semiannual Refresher Course in Laryngology, Rhinology and Otology, Sept. 24 through Sept. 29, 1945, at the College, in Chicago. The course is intensive and largely didactic, but some clinical instruction is also provided.

It is especially suited to specialists unable to devote a longer period for advanced instruction and to others seeking a comprehensive review of the field of otorhinolaryngology. The number of registrants will be limited. It is, therefore, desirable to apply for registration immediately. The fee is $50. When applying, give full details as to school and year of graduation, postgraduate training, college degrees, etc. Write to Dr. A. R. Hollender, Chairman, Refresher Course Committee, Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Ill.

PHYSICS OF THE CONDUCTION APPARATUS.*†

H. B. PERLMAN, M.D.,
Chicago, Ill.

The conduction apparatus is a complicated mechanical acoustic system whose exact function is not clearly understood. Common failure of this mechanism continues to stimulate the otologist to obtain a better understanding of its normal function. We seek ultimately to understand the factors that control its particular manner of response to sound waves, *i.e.*, the frequency response range, sensitivity and distortion and how these factors are altered in disease.

The founders of otology some 80 years ago gained a great deal of information about the conduction apparatus. They made direct observations on fixed and fresh preparations. In recent years this investigation has been resumed, stimulated by newer knowledge of acoustics and improved experimental methods.[1,2,3]

The elements of the conducting mechanism in a fresh temporal bone are movable and resilient. Their elastic properties are similar to those of the living ear. It is possible to maintain these properties for several weeks by keeping the excised temporal bone in a moist chamber. Decomposition of the soft parts is prevented by a weak aqueous merthiolate solution. The acoustic movement of the part to be studied can be detected by fastening a minute mirror to it. These mirrors measure 0.015 x 0.03 inches and have negligible mass. They are even small enough to mount on the stapes footplate, covering only about one-third of this surface. With care, many different parts of the conducting mechanism can be exposed for study (see Figs. 1 and 2). A glass tube is sealed into the external canal of the preparation with plaster of Paris and stimuli are delivered through rubber tubing connected to this glass tube. Acoustic stimuli are generated by an audiooscillator and a loud speaker driving unit connected to the prepa-

*From the Department of Surgery (Division of Otolaryngology), the University of Chicago.

†This work was aided by grants from the Douglas Smith Foundation for Medical Research of the University of Chicago.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 21, 1945.

ration in this way. For illumination of the mirror a carbon arc light is used. The reflected light point is recorded on moving film. With the mirror only a few millimeters from the center of rotation, and the movement studied about 1 meter away — optical amplification of several hundred times is obtained. With such amplification, only a moderate

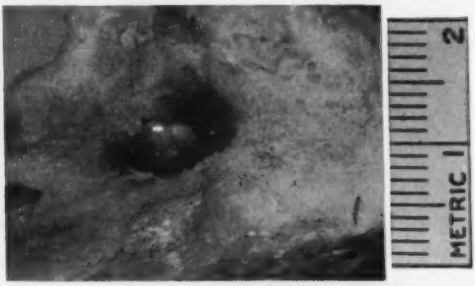


Fig. 1. Photograph of a fresh temporal bone to show the relative size of the opening in the middle fossa plate and the size of the mirror fastened to the head of the malleus for studying its motion. The mirror actually measures 0.015 to 0.3 inches.

Fig. 2. Photograph of a fresh temporal bone prepared to expose the vestibular aspect of the stapes footplate and illustrating the relative size of the attached mirror — used to study motion of the stapes.

sound stimulus is needed to obtain visible oscillation of the conducting elements. Using this general experimental equipment (see Fig. 3), a number of different observations were carried out. The conducting elements were displaced by a continuous sound, a shock wave, a small hook and by slow pressure changes produced by hand with a syringe.

Resonance Frequency: To find the resonance frequency of the intact ossicular chain, the chain was displaced by a tiny hook on the incus introduced through a hole in the middle fossa plate. A fine wire from the hook was fastened to the adjacent bone. A record was made on film of the movement of a mirror on the head of the malleus, after cutting the

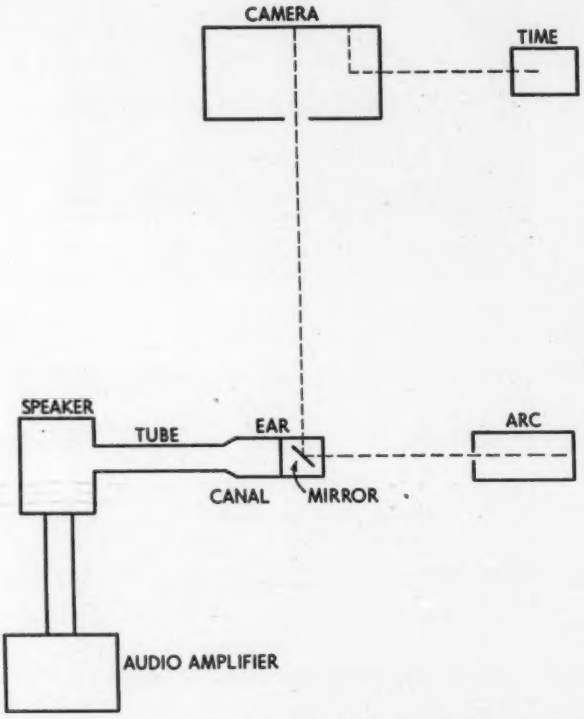
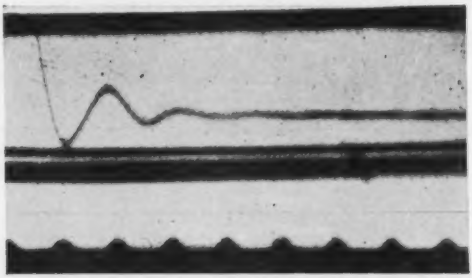
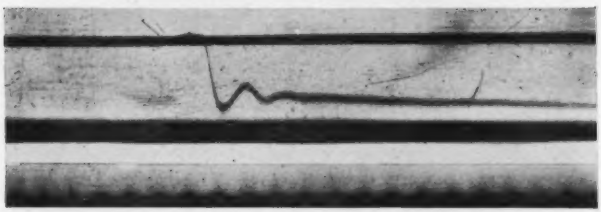


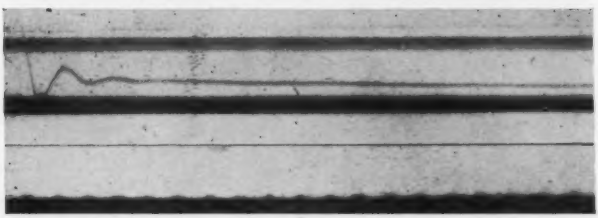
Fig. 3. Diagram of the experimental arrangement for recording acoustic movements of the conduction apparatus in a fresh temporal bone.

wire. This movement represents the inherent frequency of the chain (see Figs. 4, 5, 6, 7, 8 and 9). Another method used was to displace the chain by a shock pulse from a .32-caliber cartridge. This gave slightly different results (see Figs. 10 and 11). The first method was probably the most correct, the only factor of error being the mass of the tiny hook. This mass was negligible and when the wire was cut became partly

freed from the incus. The latter method of stimulation gave higher frequency results. The reason for this is the nature of the stimulus used. The shock wave stimulus is very rapid as well as asymmetrical, the major portion enduring about





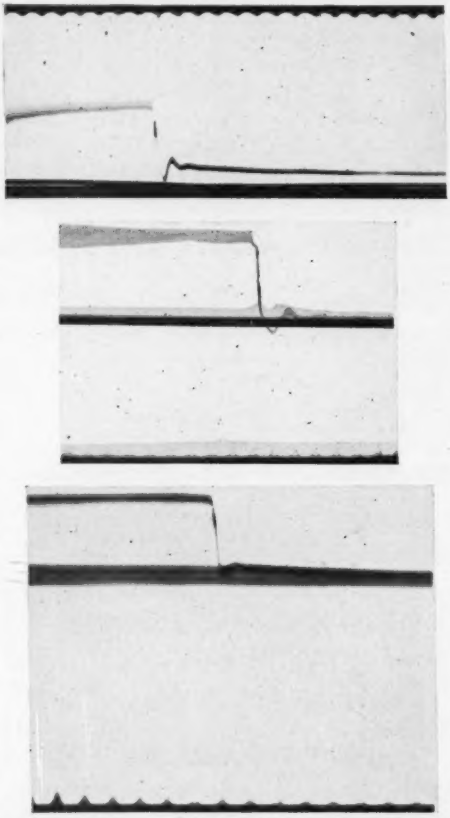


Figs. 4, 5 and 6. Series of records showing the resonance frequency and damping characteristics of the intact conducting mechanism. The intact chain was displaced towards the negative side of center with a sma l hook on the incus. The wire holding this hook was cut and the record shows the return of the malleus towards a resting position at its own speed and frequency. Timing wave 1,000 cycles per second.

Note: The displacement exceeded somewhat the elastic limits of the chain.

$^1/_{2000}$ second. The chain endeavors to follow this movement and thus a more rapid oscillation occurs. The resonance frequency obtained with the first method was around 700 cycles per second. with the second method about 1,000 cycles per second. To avoid amplitude distortion, the resonance fre-

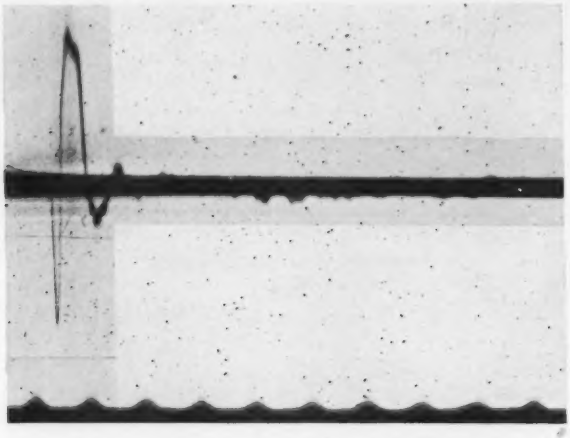
quency of a sound recording system should be several times the highest frequency to be recorded. (In the human ear this would be about 40 to 60,000 cycles per second.) The conduction system is not constructed in this way.

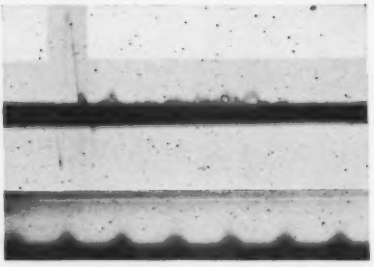


Figs. 7, 8 and 9. Records similar to those in Figs. 4, 5 and 6. The lower two indicate displacement within the elastic limits of the conducting apparatus and prompt return to the zero position. The timing wave is 1,000 cycles per second. The relatively low resonance frequency (about 700 cycles per second) and the high degree of damping (passing but once beyond the midposition before coming to rest) is noteworthy.

The resonance frequency of the conduction apparatus lies within the frequency range of response, but amplitude distortion is prevented by increasing the resistance or damping of this anatomical unit.

Damping Factor: The speed with which the conducting elements return to rest after being displaced describes their damping or resistance. This is an important property in a sound-recording system. A system with little damping continues to oscillate as its own frequency for a considerable time. This would be obviously undesirable for the ear. The





Figs. 10 and 11. Records of movement of the malleus when the intact chain is stimulated by a single shock or blast wave. The high degree of damping is again evident. A higher frequency of oscillation than in Figs. 4 to 9 is due to the frequency of the stimulus.

conducting elements rapidly return to rest after displacement; that is, the damping or resistance is large. Some of the records that were obtained in the experiments on the resonance frequency were adequate for analysis of the damping factor. Critical damping is a term used to describe the degree of damping when a system comes back from its dis-

placed position to the position of rest without passing this position of rest. The human conducing elements are not critically damped, since they are found to oscillate a few cycles before coming to rest. By determining the ratio of these wave crests, the damping factor can be determined. Its position in Fig. 12 indicates approximately the amount of amplitude distortion of the human conducting system as related to the resonance frequency of the conduction apparatus as a whole. This small amount of distortion due to the lack of critical resistance or damping of the middle ear mechanism is probably completely removed by the fluids in the cochlea. The induced movement of the end-organ is further damped

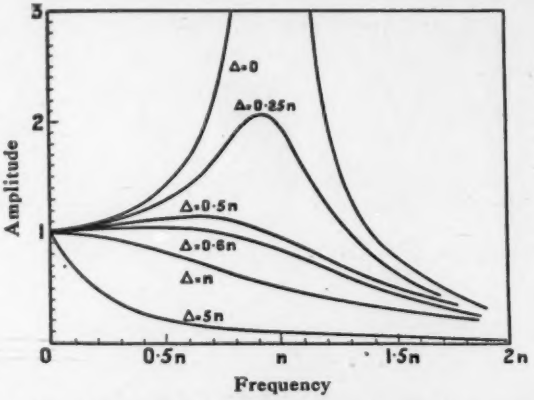


Fig. 12. Forced vibration. Relation between frequency and amplitude of displacement for various degrees of damping (from Davis, A. H.: Modern Acoustics. New York, The Macmillan Co., 1934). At the malleus, damping (△) is between 0.5 and 0.25 n.

because the movement occurs in liquid instead of in air. Critical damping is therefore probably achieved for the end-organ of the cochlea.

Mass: It is difficult to define the effective mass of the conducting apparatus. The actual weight of the component parts of this apparatus does not represent the effective mass of this system with respect to its response to sound waves. This mass is modified by the particular suspension of the ossicular chain and its manner of movement. A gross inspection of the moving chain either in response to slow pressure changes or to the pressure changes of sound waves indicates that the

malleus and incus rock around an axis extending from the anterior ligament of the malleus to the fan-shaped ligament of the incus. This axis is so placed as to apparently balance out the mass of the drum, malleus and incus. Hence these units are able to respond to stimuli of small intensity. With the experimental equipment used, allowing an optical mag-

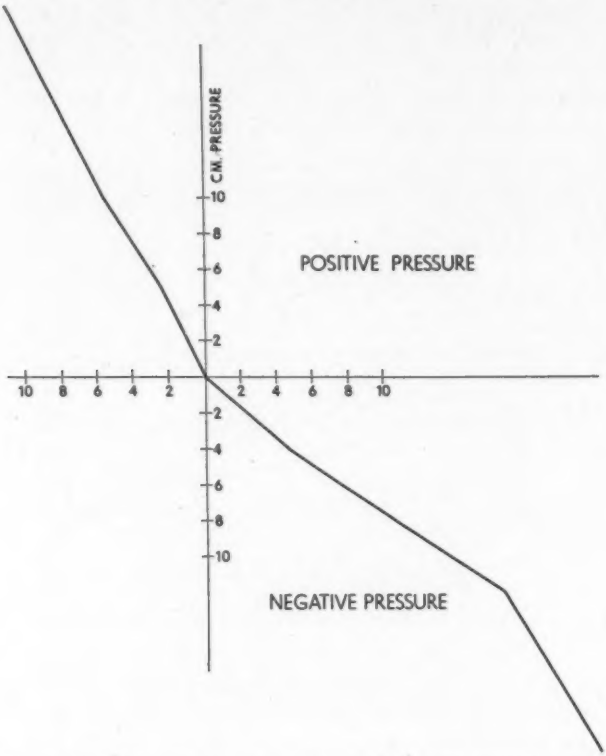


Fig. 13. Displacement of head of malleus (in mm.) to static pressure (in cm. H_2O). Amplification factor about 200.

nification of about 200, movement of the head of the malleus was observed to sounds of only moderate intensity — about 50 db. above threshold. It is reasonable to consider that with greater magnification movement to smaller intensities may be seen. On the other hand, Dahmann considers that the chain moves in and out as one unit for sounds of small inten-

sity and undergo rocking movement only to very loud sounds. Change in the position of the axis of rotation with very great sounds may alter the effective mass of the chain and thus further reduce the amplitude of motion. For all intensities

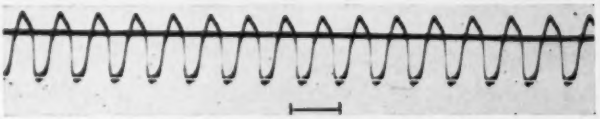


Fig. 14. Acoustic oscillations of malleus. Note asymmetry of movement about center. Negative wave is below base line.

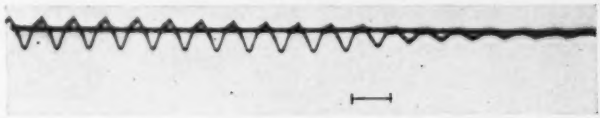
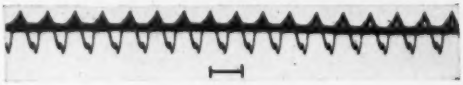


Fig. 15. Oscillation of the malleus to a sound of decreasing intensity, showing that its movement may be all negative of center with more physiologic stimuli.



Figs. 16. Oscillations of the stapes footplate to a low frequency impure sound.

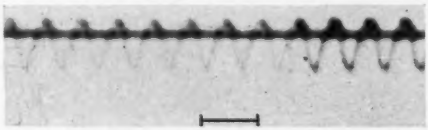


Fig. 17. Acoustic oscillation of the stapes footplate to strong low frequency sound. Note major oscillation is negative of center (below).

Fig. 18. Stapes oscillations to sound of decreasing intensity.
Note for Figs. 14 to 18: Bracketed line represents 10 milliseconds.

used in this experiment no change in axis with change in intensity could be determined. This was also true for the stapes footplate, although Bekesy claims that this axis changes 90°. The stapes moved from an axis near its posterior attachment.

The particular manner of suspending the malleus, incus and drum to place the axis of rotation through the center of this mass is indeed an excellent solution to the problem of

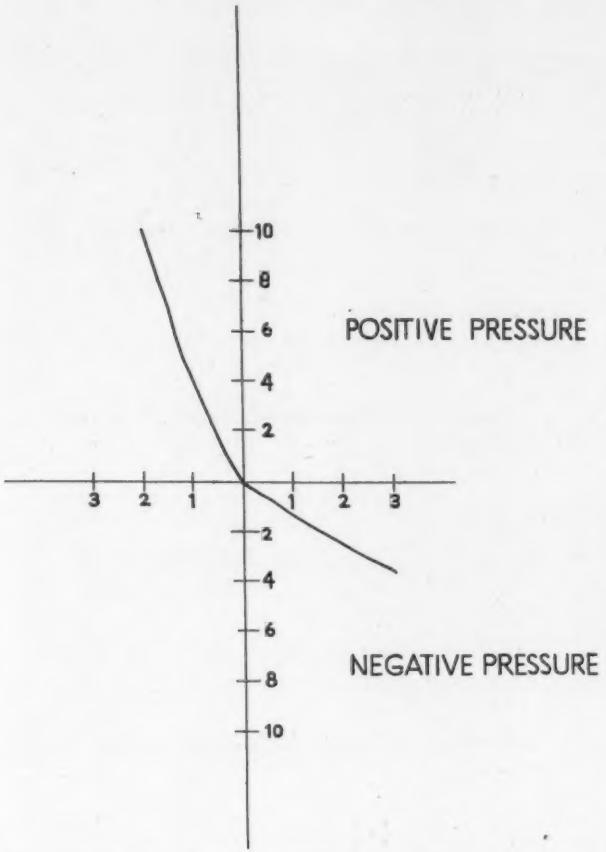


Fig. 19. Displacement in millimeters of a light point from a mirror on the stapes footplate to positive and negative static pressure in centimeters of water.

obtaining acoustic sensitivity with relatively large anatomical units.

Distortion: The conducting mechanism does not move with equal facility about its resting position.[4] It moves more read-

ily towards the negative than towards the positive side of center. This is particularly true of the intact chain, but also for the chain disconnected from the stapes. The drum and malleus preparation alone retains elasticity and moves more easily toward the negative side of center. This asymmetry is present for acoustic as well as for static pressure changes (see Fig. 13). Asymmetry of oscillation about center introduces distortion of the incoming stimulus (see Figs. 14 and 15). The stapes shows the most marked asymmetry—vibrating mostly on the negative side of center (see Figs. 16, 17, 18 and 19). Accentuation of the asymmetry of movement of the stapes over the malleus indicates a certain amount of differential movement at the incudomalleolar and incudostapedial articulation. This may in part be a protective mechanism against great positive pressure. The negative half of the incoming sound wave appears to be the effective stimulus to the cochlea. This is in line with other observations on cochlear microphonics that indicate that the microphonic is developed with the outward movement of the stapes and inward movement of the round window membrane.[5] The stapes resists inward motion so that positive pressure delivered to it is spent in differential movement between the ossicles; *i.e.*, of the malleus at the incudomalleolar joint and the incus at the incudostapedial articulation. For outward movement or negative pressures the stapes follows more faithfully the movement of the incus and malleus, hence no differential movements at the joints.

Middle Ear Muscles: The effect on the acoustic oscillation of the chain of contraction of the middle ear muscles can be studied by tying threads on the exposed tendon or muscle and pulling gently in the direction of physiologic contraction. The contraction not only reduces the amplitude of acoustic oscillation but reduces this asymmetrically. It moves the base line or axis of rotation medially. The negative phase of the oscillation is more affected than the positive phase. The pull of the tensor is in such a direction as to limit or interfere with the outward movement of the malleus; *i.e.*, the negative phase of the oscillation. Similarly, the pull of the stapedius is such as to tend to force the stapes into the labyrinth and when the incus is gone, such an inward motion actually takes place, there being enough elasticity of the annular ligament to permit this. This direction of force interferes with the

outward displacement of the stapes; *i.e.*, the response to the important effective negative phase of the sound wave.

Summary: Some of the physical properties of the human conducting mechanism were determined on fresh temporal bone. Studies along these lines can help the otologist to understand how the normal conducting mechanism functions as an acoustic instrument and the particular manner of its response to sound waves. The significance of these properties in determining the response of the middle ear structures to sound waves is pointed out.

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950 East 59th Street.

ENDAURAL MASTOIDECTOMY — FIVE YEARS' EXPERIENCE.*

GORDON J. MCCURDY, M.D.,
Providence, R. I.

Since mastoidectomy through the endaural incision was first described by Lempert in 1928,[1] and further described by him in 1938,[2] there have been a number of articles published about it, some praising and one condemning it.[3,4,5,7] The majority opinion seems to be that it is a good method for radical mastoidectomies where the majority of work is in the middle ear, attic and antrum, but that it is not the method of choice in complete, or simple, mastoidectomies for acute mastoiditis. I have used the endaural approach in all of my temporal bone surgery since early in 1939, when I was taught the method by Dr. Lempert incidental to learning the fenestration operation, and have used it ever since.

It is not going to be argued here that all operators should perform their mastoidectomies by the endaural route, for adequate and complete work can be done by either the endaural or postaural method. What is proposed is to show the advantages and disadvantages as I have seen them in my experience during the past five years.

TECHNIQUE.

A complete description of the technique is not necessary. It has been adequately dealt with by Lempert[2] and by Wishart.[4] Both lay great stress on the difficulty and great exactitude necessary in making the incision. Both make it sound much more difficult than it is. The following description is an attempt to simplify it. It can be done by anyone, easily. Some practice on a cadaver is helpful.

The incisions are best made with Lempert's spear-shaped knife or perforator; they can, however, be made with any small scalpel. They are made in the posterior and superior

*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1945.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 1, 1945.

part of the ear canal in the mobile section just within the cartilage of the auricle. None of the cartilage is injured, and none of the attachments of the auricle are cut, so that there can be no drooping or collapsing of the ear canal afterwards, as is sometimes seen in cases operated upon postauricularly.

The first incision is in the posterior wall of the canal, starting from above at the junction of the bony canal wall and the membranous part, and going down and out diagonally to the lower border of the conchal cartilage. The second is from the beginning of the first incision, upwards, just anterior to the helix, and is thus continued outside of the ear canal. This is made to the bone up to the edge of the temporal muscle, and then the muscle is spared and the subcutaneous tissues incised to the temporal fascia. This point is important. The Lempert perfortor aids here. The length of this incision varies with the case, depending on the size of the auricle and of the mastoid. It can be lengthened at will to give more room for stretching. The third incision connects the lower end of the first to the upper end of the second and is curved, just skimming the anterior edge of the concha. The first two are perpendicular to the surface of the bone. The third is pointing backward at an angle, undercutting the auricle, so that the amount of periosteum removed is greater than the amount of skin. This is essential and important in order not to be impeded by thick subcutaneous tissue while working posteriorly on the mastoid. This whole area is removed and discarded, as it is not needed or wanted in the closure. There is nothing intricate or difficult about the incision as Wishart claims.[4] If done as outlined here, with the posterior incision undercutting the skin, and with the vertical incision sparing the temporal muscle, it is completely adequate and very easy.

Lempert modifies the incision for radical operations by starting the first and second incisions at the eardrum. In my opinion, however, the incisions as outlined above for the simple mastoidectomy are adequate for all cases. The skin of the ear canal can then be utilized in making any kind of flap desired in closing, such as the ones mentioned by Shambaugh,[8,9] Lathrop,[10] etc. Thus, the flap can be made to fit the case. I do not care for the one described by Kettel,[6] as it uses the portion usually discarded as a plastic flap in the inferior

of the wound. This part contains hair and sebaceous glands and is not needed anyway.

Removal of the triangle of skin reveals the cortex of the mastoid quite adequately. A periosteal elevator is used to undermine the skin as far down the mastoid process as needed, or as far anteriorly or posteriorly.

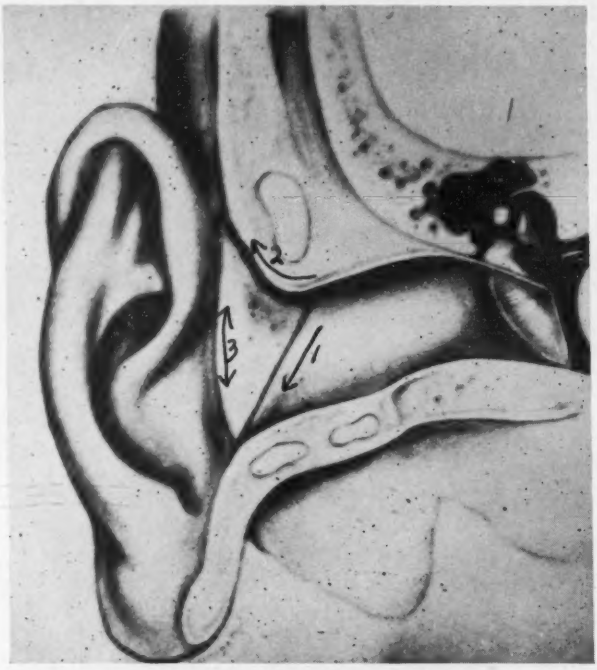

Fig. 1. The incision, after Lempert.

The next point in the technique is the point of opening of the bone. As described by Lempert, it is at the junction of the middle and inner third of a line between the spine of Henle and the superoposterior part of the eardrum. As he actually uses it and as it is usually made, the entrance point is nearer Henle's spine. The burr is driven into the antrum, or cells near it, and then the cortex removed with burrs, rongeurs and curettes. A few operators use gauges and mallets, as in the postaural method.

From here on, the operation is carried out as in any mastoidectomy. A complete, simple, modified radical, or radical, or any of the more extensive procedures is done just as postauricularly. The use of long-handled curettes is of advantage. A little different technique of using them is necessary than in a postauricular incision, but when once learned (and it is simple), it will be found helpful in any bone surgery.

Figs. 2, 3, 4, and 5 show an ear before, during and after a modified radical operation. They show how adequate the exposure is.

Fig. 2. Ear before operation.

In performing the mastoidectomy, certain areas are, of course, easier than others. The main body of the mastoid is usually not difficult. The approach to the antrum is a little more direct than postaurally. Occasionally, when the posterosuperior cells extend backwards a long distance, some difficulty is obtained in using a rongeur to remove the cortex. Electric burrs usually obviate this. In some mastoids with a small auricle placed high in relation to the tip, the tip cells

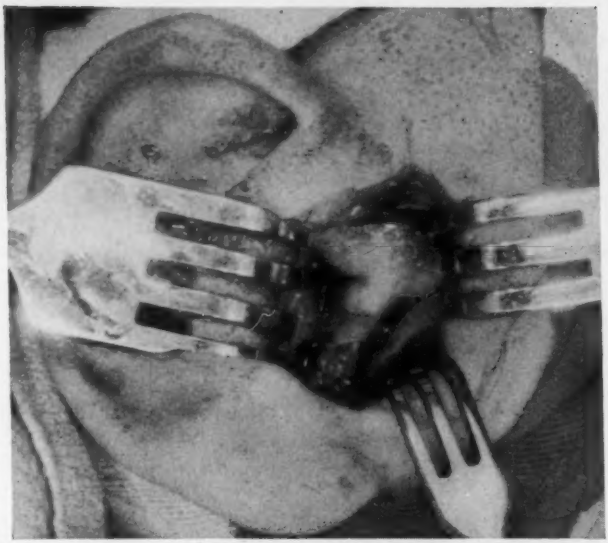

Fig. 3. Exposure of cortex showing old perforation as a result of an incompletely operated simple mastoidectomy in childhood.

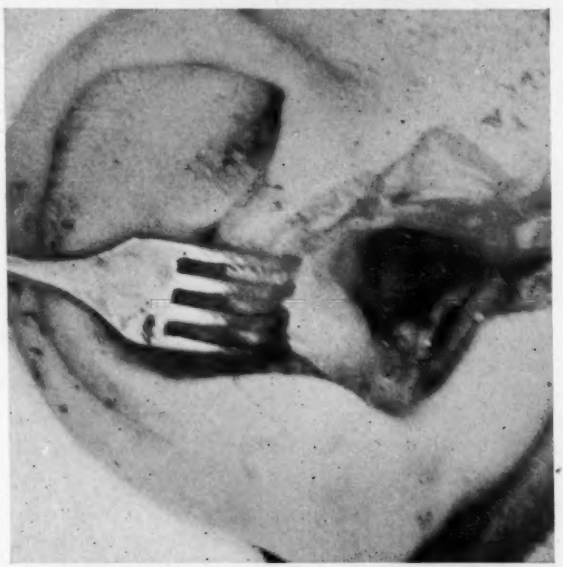

Fig. 4. Completely operated modified radical cavity.

are hard to exenterate, but the mobile endaural window can be stretched so that these cells do not present too much of an obstacle. The sinus plate area and the main mesioposterior group of cells as described by Meltzer[11] are not more difficult than postaurally nor is the delineation of the digastric crest.

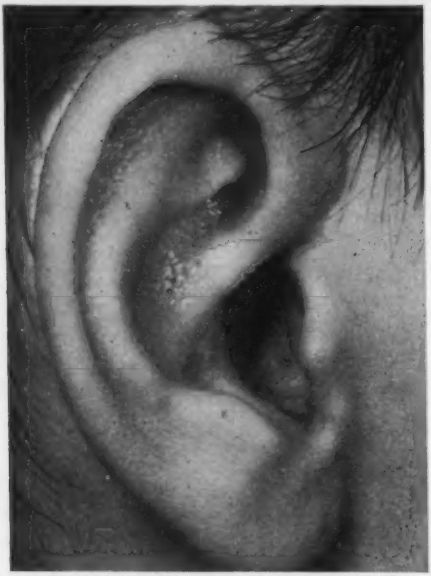

Fig. 5. Healed incision eight weeks after operation.

The retrofacial cells usually are easier than in the postaural approach. The direct view makes one feel much more secure in this region. The hardest place to get at is the group of cells behind the sinus plate in those cases where the sigmoid sinus makes its bend under the antrum or where it is very superficial close to the ear canal. It is not the distance here so much as the angle of approach to the cells that is difficult. In these cases a postauricular incision can be added and then sutured again tightly at the end. As the endaural window adequately drains the area, no postaural drainage opening is needed. The percentage of cases where the extra incision is needed is extremely small, only two needing it in this series.

During the last five years I have operated upon 122 mastoids at the Rhode Island Hospital, both ward and private. These cases are summarized in the following tables.

TABLE 1. DATA ON CASES OF ENDAURAL SIMPLE MASTOIDECTOMIES.

Case No.	Initials	Age, Yr.	Date of Operation	Date of Discharge	History	X-ray Findings	Result
1	M. J.	5	3/27/39	4/ 2/39	Pain; discharge 3 wks.; postauricular edema; tenderness, drooping of canal.	Cloudiness; breaking down.	Healed dry.
2	W. A.	14 mos.	5/ 1/39	5/16/39	Acute mastoiditis 6 days.	Destruction.	Healed dry.
3	J. D.	16	5/16/39	5/19/39	Pain and discharge few days 6 mos. ago; dried up; no pain except on bumping head; ear began to stick out 2 wks. ago; gradually developed large postaural swelling; Bezold abscess.	Large cells to petrous tip; destruction.	Healed dry.
4	J. O.	11	5/25/39	6/ 1/39	Cold 2 wks. ago; pain in ear; spontaneous rupture.	None.	Healed dry.
5	L. C.	24	6/ 5/39	6/12/39	Foul discharge 2 mos.; pain; tenderness; central perforation.	Area of infection.	Dry ear; healed perforation.
6	L. P.	11 mos.	6/ 9/39	6/19/39	Discharge 3 mos.; polyp on posterosuperior canal wall.	Clouding.	Unknown.
7	A. B.	14	6/10/39	6/17/39	Discharge, dizziness and vomiting 3 wks.	Destruction.	Healed dry.
8	J. F.	14 mos.	6/13/39	6/30/39	Cold 1 wk.; redness and postaural swelling 4 days; spontaneous perforation of drum.	None.	Developed bronchopneumonia; discharge from other ear; sulfapyridine; both ears dry.
9	P. D.	53	7/ 3/39	7/ 8/39	Pain and discharge 3 mos.; perforation of drum.	Destruction.	Healed dry.
10	J. H.	24	10/ 9/39	10/12/39	Discharge 10 wks.	Destruction.	Healed dry.
11	E. S.	3	1/ 5/40	1/10/40	Acute ear 3 wks.; clinical signs of mastoiditis.	Destruction.	Healed dry in 2 wks.
12	G. A.	11	2/13/40	2/20/40	Acute mastoiditis; staphylococcus empyema.	Clouding; destruction.	Healed dry.
13	J. R.	48	4/11/40	4/27/40	Otitis media 1 wk.; headache, tinnitus; vomiting.	Destruction.	Healed dry.
14	J. R.	48	4/19/40	4/27/40	Otitis media 1 wk.; headache; tinnitus; vomiting.	Destruction.	Healed dry.
15	J. S.	15	4/26/40	4/30/40	Earache; dizziness.	Haziness.	Healed dry.

(Continued)

TABLE 1. DATA ON CASES OF ENDAURAL SIMPLE MASTOIDECTOMIES (Continued).

Case No.	Initials	Age, Yr.	Date of Operation	Date of Discharge	History	X-ray Findings	Result
16	A. R.	29	5/ 7/40	5/11/40	Draining ear 2 mos. with tinnitus, vertigo, deafness; otosclerosis of opposite ear.	Sclerosis; haziness.	Continued discharge; revision; dry after X-ray therapy to Eustachian tube and nasopharynx.
17	H. H.	27	9/ 9/40	9/13/40	Discharge 6 mos.; headache; vertigo; anteroinferior perforation.	Destruction.	Continued discharge; vertigo; revision; dry ear; tuberculosis found later.
18	E. T.	51	1/21/41	1/25/41	Subacute ear 6 wks.	Destruction.	Healed dry.
19	A. M.	67	3/ 1/41	3/ 3/41	Discharge 1 mo.	Destruction.	Healed dry.
20	L. F.	53	5/ 5/41	5/13/41	Postaural headache; intermittent pain; slight fever.	Sclerosis; breaking down.	Healed dry.
21	A. W.	53	5/15/41	5/22/41	Pain and discharge 4 wks; postaural tenderness; bulging drum.	Destruction.	Healed dry.
22	A. M.	82	5/24/41	5/29/41	Pain and discharge 6 wks.; tenderness; perforation; diabetes.	Destruction.	Healed dry.
23	J. B.	8	7/11/41	7/20/41	Discharge 10 days; postaural swelling.	Destruction.	Healed dry.
24	E. B.	13	10/10/41	10/16/41	Foul discharge and swelling 7 days; stiff neck; tenderness.	Destruction.	Healed dry.
25	C. D.	14	3/10/42	3/16/42	Postaural swelling and tenderness; upper respiratory infection 5 wks. ago.	Destruction.	Healed dry.
26	J. D.	2	3/19/42	3/24/42	Postauricular swelling 3 days; red drum; tonsillitis 2 mos. ago.	Destruction.	Healed dry.
27	J. D.	7	5/16/42	5/22/42	Pain 2 days; tenderness; thick discharge; measles 4 wks. ago.	Destruction.	Healed dry.
28	A. B.	70	7/15/42	7/23/42	Pain 2 wks.; dizziness; headaches; foul discharge; diabetes.	Obliteration of cell outline.	Healed dry.
29	R. C.	13	8/15/42	8/19/42	Discharge 3 wks.	Destruction.	Healed dry.
30	E. A.	46	1/24/43	2/ 8/43	Pain and discharge 3 wks.	Destruction.	Continued discharge; nonhealing; revised 2/2/42; postaural revision 3/1/43 because of spontaneous perforation; revised 8/23/43; healed dry.
31	C. C.	24	1/24/43	1/30/43	Earache 3 days; postaural edema; tenderness; pulsating discharge.	Clouding; decalcification.	Healed dry.

(Continued)

TABLE 1. DATA ON CASES OF ENDAURAL SIMPLE MASTOIDECTOMIES (Continued).

Case No.	Initials	Age, Yr.	Date of Operation	Date of Discharge	History	X-ray Findings	Result
32	E. G.	68	5/ 3/43	5/22/43	Acute mastoiditis; syphilis.	Complete destruction.	Healed dry.
33	P. P.	6	6/ 2/43	6/ 9/43	Intermittent pain 2½ yrs.; discharge 6 wks.	Destruction.	Continued discharge; modified radical revision 6/25/43; healed dry.
34	E. W.	57	12/17/43	1/ 4/44	Pain 10 days; swelling; tenderness; no discharge; deafness; heart difficulty; cold 1 mo. ago.	Destruction.	Healed dry.
35	M. H.	9	1/ 8/44	1/20/44	Measles; subacute bilateral mastoiditis; edema of both canals.	Bilateral destruction.	Both ears healed dry.
36	M. H.	9	1/17/44	1/20/44			
37	C. D.	3	1/17/44	1/22/44	Tenderness and discharge 6 wks.	Clouding; decalcification.	Healed dry.
38	C. C.	6	1/19/44	1/19/44	Discharge and tenderness 1 wk.	Clouding; decalcification.	Healed dry.
39	A. M.	27	1/21/44	1/26/44	Earache and discharge 3 wks.; tenderness; edema.	Destruction.	Healed dry.
40	W. W.	12	2/ 6/44	2/15/44	Acute mastoiditis 1 wk.; tenderness over mastoid.	Destruction.	Healed dry.
41	J. C.	20	3/27/44	3/31/44	Acute mastoiditis; discharge; tenderness.	Clouding; destruction.	Healed dry.
42	E. C.	28	3/28/44	3/31/44	Acute mastoiditis 3 wks.; discharge; tenderness.	Sclerosis; clouding.	Healed dry.
43	J. R.	67	3/29/44	4/ 2/44	Acute mastoiditis 3 wks.; discharge; tenderness.	Destruction.	Healed dry.
44	M. A.	16	4/10/44	4/15/44	Acute mastoiditis 7 wks.; tenderness; discharge.	Destruction; clouding.	Continued discharge; revision 5/12/44; healed dry.
45	S. M.	49	6/22/44	6/26/44	Vertigo; tinnitus; deafness; discharge which stopped.	Decalcification.	Healed dry; poor hearing and tinnitus still present; vertigo gone.
46	G. L.	13	7/ 8/44	7/19/44	Pain 2 wks.; high fever; small perforation of drum; no discharge; has meningism.	Destruction.	Healed dry 2 wks.

TABLE 2. DATA ON CASES OF ENDAURAL RADICAL MASTOIDECTOMIES.

Case No.	Initials	Age, Yr.	Date of Operation	Date of Discharge	History	X-ray Findings	Result
1	E. F.	17	2/20/39	2/26/39	Chronic mastoiditis; pain; vertigo.	None.	Healed dry.
2	W. I.	12	5/ 5/39	5/13/39	Chronic ear; vertigo.	None.	Continued discharge; revision 7/11/39; ear ran; revision 2/13/40; dry ear finally after X-ray therapy to auditory tube and nasopharynx.
3	W. S.	9	6/ 8/39	6/12/39	Chronic ear with cholesteatoma; anorrhexia; headache; erosion.	Sclerosis.	Continued discharge; revisions 10/17/39 and 2/20/40; discharge less but recurred; revision 7/16/40; healed dry.
4	R. M.	15	6/20/39	6/24/39	Intermittent ear discharge 10 yrs.; posteroinferior perforation of drum.	Lost.	Healed dry.
5	J. R.	32	7/25/39	7/28/39	Chronic ear 10 yrs.; vertigo 5 days; arrested tuberculosis.	Sclerosis; destruction.	Patient lost sight of.
6	P. J.	48	2/ 3/40	2/17/40	Chronic ear 1 yr.	None.	Healed dry.
7	E. F.	17	4/19/40	4/24/40	Chronic mastoiditis; vertigo.	None.	Healed dry.
8	H. S.	45	4/24/40	4/30/40	Chronic ear 30 yrs.; vertigo 2½ mos.	None.	Healed dry.
9	H. S.	12	4/30/40	5/ 4/40	Chronic ear; previous simple mastoidectomy unsuccessful.	None.	Healed dry but became reinfected later.
10	D. D.	38	5/22/40	5/27/40	Chronic ear many years since early scarlet.	Rarefied area.	Healed dry.
11	D. S.	9	6/11/40	6/19/40	Chronic ear 8 yrs.	Sclerosis.	Healed dry.
12	A. U.	20	8/26/40	8/30/40	Chronic ear with foul discharge 5 yrs.	Sclerosis.	Small area of suppuration in middle ear; to revise.
13	R. C.	22	8/27/40	9/ 1/40	Chronic ear; postaural radical 2 yrs. ago; did not come for dressings; did not heal.	No cells present.	Patient again failed to carry on treatment; result unknown.

(Continued)

TABLE 2. DATA ON CASES OF ENDAURAL RADICAL MASTOIDECTOMIES (Continued).

Case No.	Initials	Age, Yr.	Date of Operation	Date of Discharge	History	X-ray Findings	Result
14	M. V.	29	9/22/40	9/26/40	Discharging ear and vertigo 18 yrs.	Sclerosis.	Healed dry.
15	H. Z.	27	12/26/40	12/31/40	Chronic ear.	Sclerosis.	Healed dry.
16	E. B.	30	3/25/41	4/ 5/41	Discharging ear 3 yrs; postaural tenderness; posterior perforation of drum.	None.	Healed dry.
17	A. C.	29	4/ 7/41	4/11/41	Recurrent mastoiditis; operation 1 yr. ago; destroyed eardrum.	None.	Healed dry.
18	J. S.	9	6/14/41	6/18/41	Discharge 7 yrs.; destruction of eardrum; cholesteatoma.	Sclerosis.	Healed dry.
19	P. V.	14	7/ 1/41	7/ 5/41	Discharge 12 yrs.; deafness; destruction of drum.	Sclerosis.	Healed dry; improved hearing.
20	W. K.	18	7/12/41	7/16/41	Discharge 15 yrs.; chronic sinusitis and allergy.	Clouding; destruction.	Continued discharge; revision 1/3/42; middle ear remained moist.
21	J. C.	30	8/ 4/41	8/ 9/41	Discharge 20 yrs.; absent drum.	None.	Healed dry.
22	D. H.	20	9/ 3/41	9/ 7/41	Chronic bilateral running ears; loss of hearing; large posterior perforation of both drums.	Sclerosis.	Healed dry.
23	N. B.	18	10/ 6/41	10/ 9/41	Discharge all his life; destroyed drum.	Sclerosis.	Healed dry.
24	A. B.	31	11/22/41	11/29/41	Discharging rt. ear 15 yrs.; pain 2 wks.; rt. facial paralysis 1 wk.; tenderness; cholesteatoma; erosion of superior portion of canal.	Sclerosis.	Healed dry; recovery from facial paralysis.
25	F. L.	25	1/ 8/42	1/12/42	Pain; discharge 1 yr.; destroyed eardrum.	Sclerosis.	Healed dry.
26	M. N.	37	1/12/42	1/16/42	Intermittent discharge and pain 18 yrs.; recent dizziness; destruction of drum.	Sclerosis.	Healed dry.

(Continued)

TABLE 2. DATA ON CASES OF ENDAURAL RADICAL MASTOIDECTOMIES (Continued).

Case No.	Initials	Age, Yr.	Date of Operation	Date of Discharge	History	X-ray Findings	Result
27	R. D.	19	3/17/42	3/21/42	Chronic discharge since age of 4 mos.	Probable cholesteatoma.	Healed dry.
28	M. V.	9	5/12/42	6/ 5/42	Bilateral intermittent discharge 4 yrs.; foul discharge with narrowing of canals; polyps in both ears.	Sclerosis.	Continued discharge; revision 5/25/44; still discharging.
29	C. L.	50	6/ 2/42	6/ 9/42	Discharge 33 yrs.; dizziness 1 wk.; superior perforation of drum; fistula reaction.	Sclerosis.	Healed dry.
30	M. C.	51	6/25/42	7/ 1/42	Discharge 15 yrs.; absent drum.	Sclerosis.	Healed dry.
31	N. F.	41	6/29/42	7/ 3/42	Discharge through large perforation several yrs.; recent pain; cholesteatoma.	Sclerosis.	Healed dry.
32	E. P.	29	8/ 7/42	8/11/42	Bilateral chronic running ears.	Sclerosis.	Unknown; refused to return for treatment.
33	R. C.	43	9/19/42	9/25/42	Discharge 5 yrs.; pain and dizziness 6 mos.; posterior perforation.	Sclerosis.	Healed dry.
34	J. P.	26	1/ 4/43	1/ 8/43	Chronic discharge; destroyed drum; cholesteatoma.	Sclerosis.	Healed dry.
35	P. P.	59	5/28/43	6/ 4/43	Chronic discharge; dizziness.	Sclerosis.	Healed dry.
36	W. T.	27	7/ 1/43	7/ 3/43	Discharge 5 yrs.; recent pain.	Sclerosis.	Healed dry; has had bad exacerbations.
37	C. M.	18	*/22/43	7/29/43	Radical mastoidectomy done 4 yrs. ago; continuous discharge; pain 12 hours; postauricular swelling; incised and drained by another surgeon 8 days ago.	Sclerosis.	Unknown; patient disappeared.
38	A. B.	23	8/20/43	8/24/43	Chronic discharge; granulations.	Sclerosis.	Healed dry.
39	A. D.	52	8/30/43	9/ 2/43	Chronic discharge; recent pain; superior perforation of drum.	Sclerosis.	Healed dry.
40	E. T.	59	8/31/43	9/ 3/43	Chronic discharge; pain 2 wks.; granulations.	Sclerosis.	Healed dry.

(Continued)

TABLE 2. DATA ON CASES OF ENDAURAL RADICAL MASTOIDECTOMIES (Continued).

Case No.	Initials	Age, Yr.	Date of Operation	Date of Discharge	History	X-ray Findings	Result
41	G. M.	38	9/13/43	9/16/43	Discharge many yrs.; pain several mos.; canal full of polyps.	Sclerosis.	Healed dry.
42	A. B.	18	9/14/43	9/18/43	Chronic discharge; superior perforation.	Sclerosis; cholesteatoma.	Healed dry.
43	J. F.	32	10/20/43	10/25/43	Chronic discharge; posterosuperior perforation in ear canal; absent drum.	Sclerosis.	Healed dry.
44	F. G.	58	10/26/43	11/ 1/43	Discharge 1 yr.; dizziness several wks.; posterior superior perforation with granulations.	Sclerosis.	Healed dry.
45	T. B.	41	2/ 7/44	2/11/44	Chronic discharge; thick drum; vertigo; posterosuperior perforation.	Sclerosis.	Healed dry.
46	F. M.	54	3/29/44	3/31/44	Intermittent chronic discharge; large central perforation.	Sclerosis.	Still draining last exam.; has not returned for 4 mos.
47	M. M.	53	4/ 8/44	4/15/44	Osteoma of canal removed 10 yrs. ago; regrowth of osteoma with complete blockage of canal; labyrinthitis; facial paralysis.	Sclerosis.	Facial paralysis and labyrinthitis cleared; revision done later to enlarge canal; small spot in middle ear with granulation on it.
48	J. S.	28	4/20/44	4/24/44	Chronic discharge; large perforation.	Sclerosis.	Healed dry.
49	M. S.	47	4/28/44	5/ 1/44	Chronic discharge after childhood incomplete operation; postauricular perforation.	Operative defect.	Healed dry.
50	L. D.	18	5/ 2/44	5/ 7/44	Chronic discharge; dizziness 1½ yrs.; large inferior perforation.	Sclerosis.	Healed dry.
51	J. P.	20	6/30/44	7/ 3/44	Chronic discharge before and after endaural modified radical; perforation in attic through an otherwise well-operated looking endaural cavity.	Cholesteatoma cavity.	Healed dry.
52	E. V.	27	7/ 3/44	7/ 6/44	Chronic discharge; absent drum.	Sclerosis.	Healed dry after nasal allergy controlled.
53	H. R.	19	7/10/44	7/11/44	Chronic discharge; drum destroyed.	Sclerosis.	Healed dry.

TABLE 3. DATA ON CASES OF ENDAURAL MODIFIED RADICAL MASTOIDECTOMIES.

Case No.	Initials	Age, Yr.	Date of Operation	Date of Discharge	History	X-ray Findings	Result
1	J. P.	14	5/13/39	5/19/39	Chronic discharge; deafness.	None.	Continued discharge, revised 6/30/44; healed dry.
2	R. L.	15	5/13/39	5/20/39	Intermittent earache; discharge; vertigo on turning head; periosteal thickening; tenderness over mastoid 2 mos.; perforation in posteroinferior quadrant.	None.	Healed dry.
3	I. W.	25	4/ 2/40	4/19/40	Pain 7 days; no discharge; remains of furuncles seen in canal; facial paralysis 3 days; felt to be herpes zoster oticus, but exploratory done because of X-ray findings; destruction found in antrum area and solid angle.	Dullness and sclerosis; area of rarefaction in antrum area.	Healed dry.
4	B. M.	17	7/11/42	7/16/42	Discharge 6 mos.; nausea, dizziness and vomiting 3 wks.; perforation of posterior part of drum.	Sclerosis; destruction.	Healed dry.
5	S. B.	32	10/27/42	10/30/42	Discharge 1 yr.; central perforation; foul discharge.	Sclerosis.	Healed dry.
6	J. D.	19	2/25/43	3/ 1/43	Chronic discharge; superior perforation.	Sclerosis.	Healed dry.
7	E. C.	18	4/22/43	4/27/43	Intermittent discharge 15 yrs.; recent pain; edema; tenderness.	Sclerosis.	Healed dry.
8	A. M.	18	5/25/43	5/31/43	Foul discharge 6 yrs.; attic perforation.	Sclerosis.	Continued discharge; revised 9/21/43; healed dry 9 mos. later.
9	W E.	23	6/28/43	7/ 2/43	Discharge 13 yrs.; superior perforation.	Sclerosis.	Healed dry.
10	J. M.	13	6/29/43	7/ 3/43	Chronic discharge 2 yrs.; anterior perforation.	Sclerosis.	Continued discharge; 3 revisions; healed dry.

(Continued)

TABLE 3. DATA ON CASES OF ENDAURAL MODIFIED RADICAL MASTOIDECTOMIES (Continued).

Case No.	Initials	Age, Yr.	Date of Operation	Date of Discharge	History	X-ray Findings	Result
11	G. V.	24	7/ 6/43	7/10/43	Chronic discharge; posterior perforation; nasal allergy.	Sclerosis.	Healed dry; almost complete closure of perforation but recurrence of discharge with each recurrence of nasal allergy.
12	A. T.	8	7/20/43	7/24/43	Chronic discharge; posterior perforation; granulations in canal.	Sclerosis.	Healed dry.
13	M. M.	19	7/22/43	7/27/43	Chronic discharge; superior perforation.	Sclerosis.	Healed dry.
14	J. W.	43	8/10/43	8/15/43	Chronic foul discharge through antero-inferior perforation.	Sclerosis.	Healed dry.
15	M. E.	20	9/19/43	9/24/43	Discharge 2 yrs.; posterior perforation with granulations.	Sclerosis.	Healed dry.
16	F. B.	57	9/24/43	9/28/43	Chronic discharge; acute exacerbation; granulations.	Sclerosis.	Healed dry.
17	M. S.	67	11/30/43	12/ 4/43	Chronic discharge; recent pain; small posterior perforation.	Sclerosis.	Healed dry.
18	S. A.	46	2/ 8/44	2/12/44	Discharge 25 yrs.; recent vertigo; superior perforation with granulations.	Sclerosis.	Almost no healing; revision with complete healing; dry ear.
19	L. W.	10	2/10/44	2/13/44	Chronic mastoiditis after incomplete operation at 5 mos. of age; absent drum.	Sclerosis.	Healed dry.
20	J. T.	22	3/ 7/44	3/11/44	Bilateral discharge 5 yrs.; poor hearing; inferior perforation.	Sclerosis.	Discharge still present.
21	A. J.	47	3/20/44	3/24/44	Chronic discharge; pain; large superior perforation.	Sclerosis.	Healed dry.
22	B. G.	22	4/ 4/44	4/11/44	Chronic discharge; perforation in Shrapnell's membrane; cholesteatoma.	Sclerosis.	Healed dry.
23	E. B.	30	7/31/44	8/ 6/44	Chronic discharge; inferior perforation.	Sclerosis.	Still discharging.

The average length of hospital stay was five days; the shortest, one day; and the longest, 24 days after operation. In the first few years ether anesthesia was used. During the last two years local anesthesia was routinely used except on children. It makes the operation simpler because of less bleeding and makes it less of a shock to the system generally. The patients are usually out of bed in two days.

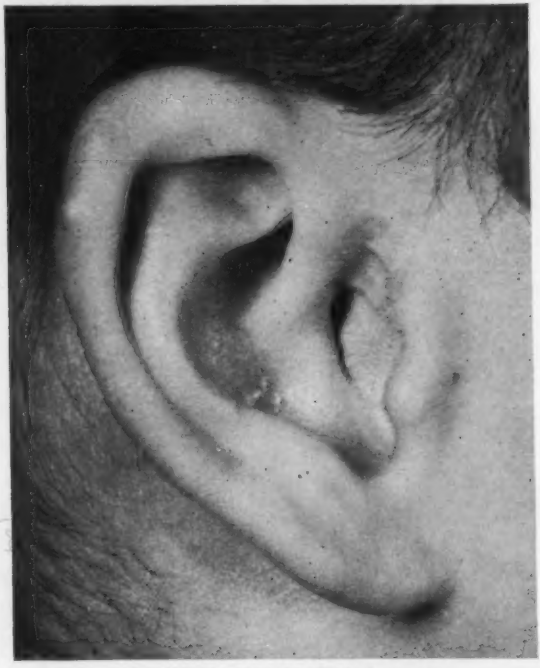

Fig. 6. Case 12, Table 1.

In this series there were no fatalities. Most patients ended with a dry ear. There were 46 complete mastoidectomies, 53 radical mastoidectomies and 23 modified radical mastoidectomies. In the radicals and modified radicals the technique was mostly that described by Lempert. In some of them the skin of the ear canal was saved and used as mentioned by Shambaugh,[8] Lathrop,[10] Woodruff and Henner,[7] etc.

DISADVANTAGES IN SIMPLE MASTOIDECTOMY.

The disadvantages found in the simple, or complete, mastoidectomies were:

1. In the early cases I felt crowded, especially in cases with a small auricle and with thickened tissues. This disadvantage disappeared when I became more familiar with the method.

2. Bleeding — In hemorrhagic type cases this has been annoying but not much more so than in postaural cases. A little blood *does* obstruct more than in the usual method.

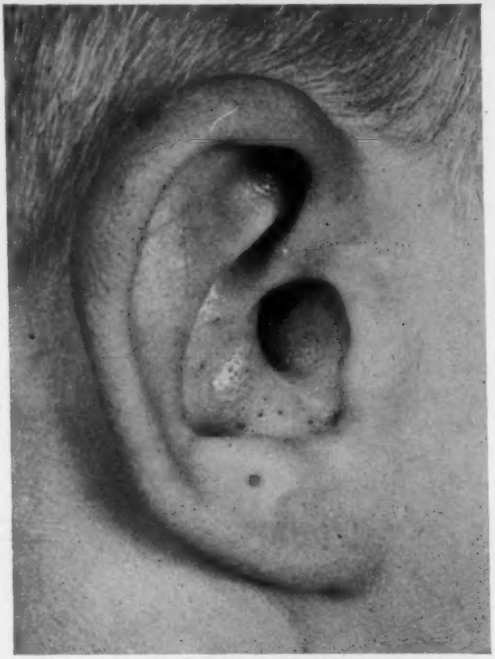

Fig. 7. Case 3, Table 2.

3. The tip cells where the tip is large are occasionally difficult. Here again, practice makes it easier.

4. If there is a high superficial knee to the sigmoid sinus with large extensions of retrosinus cells, it is hard; but there are many cases postaurally operated upon where the incision has been extended posteriorly to deal with these same cells.

5. Scar — It has been argued that the scar in front of the helix may be unsightly[4] and that there may be an atresia. So far in this series there have been three cases with poor scars that were not sightly, the worst two of which are shown in Figs. 6 and 7.

In an equal number of postaural cases there would be many more poor scars, besides drooping and collapsing of ear canals. I have never seen this latter happen in endaural incisions.

6. Time — It takes a little longer to do an endaural simple mastoidectomy; but, here again, a few more minutes (15 to

Fig. 8. Ear three days after simple mastoidectomy.

30) do not jeopardize the patient's life, and there are compensating factors to overcome this objection, as noted below.

7. Trained assistants, familiar with the operation, are of advantage. Wishart says two are needed: one to hold the retractors and one to sponge. I have found that one assistant to hold the retractors is all that is needed besides an experienced scrub nurse who is familiar with the operation.

8. Trauma to the temporal muscle is occasionally made by the points of the retractors. This is not necessary if the

retractor holder will hold them steadily. This is where a trained helper is of assistance. Occasionally there is edema of the muscle after the operation, but in only one case have I seen an abscess, and this was easily dealt with.

ADVANTAGES IN SIMPLE MASTOIDECTOMY.

The advantages in simple, or complete, mastoidectomies were:

Fig. 9. Same ear six months later.

1. There is adequate and continuous drainage during healing because of the endaural window left.

2. One can look into and see practically all the cavity as it heals. Any obstructing granulations can be curetted out. Usually there are none.

3. Zygomatic cells and the superior group of cells near the horizontal semicircular canal can be dealt with more easily as this area is perfectly exposed rather than being under the edge of a self-retaining retractor. This means better dissec-

tion of the aditus and periantral region over a large group of cases.

4. If complications develop, such as petrositis, the route of choice is through the endaural incision through the middle ear and attic and anterior to it. The petrous apex is very easily reached by the Lempert method.[12]

5. Dressings — In a few days, usually three, the dressing is removed and cotton substituted. The dressings are definitely not painful as claimed by Wishart.[4] The skin edges are usually anesthetic because some of the small branches of the auriculotemporal nerve are severed. The depths of the wound can be probed without pain.[5] There are no packs to reinsert and no messy, smelly dressings.

6. There is usually no neck stiffness from the sternomastoid muscle being detached.

7. The scar is almost invisible in most cases. Patients as a rule are proud of their scars and show them off. They are astounded at the slight disability they have. They are good advertisers. This should not be a surgical consideration, but it is pleasing nonetheless.

8. If a recurrent infection should develop in the scar tissue of the mastoid, it can be dealt with in the office with a paracentesis knife and nasal speculum with almost no pain.

9. Packing the depth of the wound is entirely unnecessary. One point in the technique might well be made here: in the first year I occasionally used to get an overriding of the skin edges, since no sutures are used; I now use a small strip of vaseline gauze in the superior incision, which is taken out the day after operation. Uniform slight edema makes the skin edges of equal height so no overriding occurs.

RADICAL AND MODIFIED RADICAL MASTOIDECTOMY.

In radical and modified radical mastoidectomies no disadvantages were noted. The advantages were:

1. The view of the middle ear and adnexa was much better and much more free from blood. In postauricular incisions

tugging on a strip of gauze to pull the auricle forward always squeezes out blood. Looking straight down is better than at an angle. One feels much safer in the region of the facial ridge and processus cochleariformis, and in taking out the tensor tympani muscle, if this latter is done.

2. The hypotympanic area and Eustachian tube are nearer and easier to work on.

3. The perilabyrinthine cells are more easily reached and better seen because the view is more direct.

4. The attic region can be cleanly dissected and the ossicles left in place in modified radicals much more easily because of the direct approach.

5. The bloody portion of the plastic on the canal is already done at the beginning of the operation, thus saving this annoyance at the end.

6. The operation lends itself to a variety of plastic skin arrangements deep in the wound that can be decided upon at the end,[6,7,8,10] because the ear canal skin can all be saved. If skin-grafting of a large area is wanted, it is just as easy as in a postauricular incision.

7. Because of the extremely good view obtained, more modified radicals will be done, thus saving more hearing in a large number of cases.

8. Packing is usually unnecessary.

9. No postauricular fistula can occur.

CONCLUSIONS.

Because of the above reasons I believe that the endaural incision is superior to the postaural in mastoid surgery.

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198 Angell Street.

SCREWWORM INFESTATION IN THE NASAL PASSAGES AND PARANASAL SINUSES.

EARL H. BROWN, M.D.,

Tucson, Ariz.

Screwworms are a serious pest of livestock. Infestation in the human is not common. It is surprising that in a cattle country such as the southwest, we do not have a higher incidence of human infestation. Screwworms in cattle diseases have been known in Texas since 1842.

One of the earliest reports* of human infestation was in 1907 by Yount and Sudler,[1] of Arizona.

The largest single number of human cases reported was by Stroud,[2] also of Arizona, in 1927.

Stewart and Boyd,[3] in 1934, suggested treatment with 15 per cent chloroform in light vegetable oil, followed by removal of the larvae with forceps.

Wallace,[4] in 1936, discussed screwworm infestation and reported a case of nasal involvement in a 72-year-old farmer. The patient was hospitalized for three weeks and treated with nasal packing saturated with benzol solution, and recovered.

Vanderslurs and Whittemore,[5] in 1938, reported a case of cutaneous larval infestation in a two-and-one-half-month-old-baby, in Minnesota. Prompt recovery followed removal of larvae with forceps.

Beachley and Bishop,[6] in 1942, reported a case of myiasis due to Bot fly larvae. Spontaneous cure followed blowing maggots from the nose. (This was not a screwworm larvae.)

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 11, 1945.

*Since reporting this case, it has been brought to my attention that the late Dr. Max A. Goldstein, of St. Louis, Mo., published an article in the December, 1897, issue of The Laryngoscope entitled, "The Texas Screwworm and Its Invasion of the Nasal Cavity." Dr. Goldstein reported a very similar case with serious involvement of the sinuses from the larvae of the macellaria type fly. It is interesting to note that Dr. Goldstein found at that time that the screwworm larvae were killed more quickly by chloroform than any other chemical.

Turnbull and Franklin[7] reported an aural infestation in a 60-year-old man who gave a history of having had a fly enter his ear canal. Maggots were removed by spraying the ear with chloroform and then irrigating. Recovery followed.

Pearman and Haseman[8] reported a case of a 58-year-old woman who had an abscess of the cheek which was opened and found to be filled with larvae.

Borey,[9] in 1943, wrote a very thorough survey of nasal myiasis. He stated that in the past 12 years previous to 1943, 21 cases of nasal myiasis had been reported in the United States. He reported five cases with one death (cause: pansinusitis with toxemia — no autopsy of the brain was done).

The screwworm is the larval stage of the cochliomyia Americana and the cochliomyia macellaria. The Americana is a carnivorous larva which lives only on healthy tissue. The macellaria lives on either living or dead tissue. The ordinary maggot seen in dead and diseased tissue is the larval stage of the blow fly. The latter lives only in dead tissue and does not destroy living tissue.

The average life cycle of the cochliomyia Americana is between two to three weeks. The female fly is attracted to some wound, by odor usually, and deposits eggs numbering 10 to 400 in shingle-like masses cemented together. Eggs of ordinary blow flies are not cemented together and are deposited in haphazard fashion. The eggs hatch in six to 21 hours and the larvae are mature in five to six days; during this maturing period they burrow into the living tissue and form pockets. After maturity the larvae begin to drop from the wound, burrow into the ground and form pupa. Seven to 14 days later the adult fly emerges. During cool weather this stage may last several months. Five to 10 days after the flies emerge they are ready to mate and lay eggs. The cycle consists of egg, larvae, pupa and fly.[10] The mature fly is a deep greenish-blue metallic color with yellow face and three dark stripes in the dorsal surface of the thorax. The egg and the pupa stage are not pertinent to this discussion. The larval stage of the screwworm has a typical appearance. The mature larva is about two-thirds inches long with approximately 12 rings or spines circling the body. This gives it the typical appearance of a screw. The head tears into living

tissue by means of a powerful oval hood surmounted by two hook-like projections. The breathing apparatus is located on the rear end.

Usually within two to three days after an infestation in the nose, the patient begins to complain of nasal occlusion, headache, sneezing accompanied by a serosanguineous discharge. The temperature may be elevated to 100° to 103°; a very disagreeable odor permeates the room from the nasal passages. The white blood count is elevated as would be expected, since considerable secondary infection takes place. Upon examination, the nasal mucous membrane is found to be greatly swollen, bleeds easily after shrinking, and masses of worms in constant motion are observed. When alive they are firmly attached and difficult to extract.

The consensus of opinion is that the best treatment consists of irrigating the nose with 50 per cent chloroform solution or spraying with straight chloroform and then removing the larvae with forceps or suction. If left untreated the worms will destroy not only the mucous membrane but also penetrate through the bone into the cranial cavity with ensuing fatal meningitis. The case herein reported barely escaped this complication, even though under treatment from the first day of onset of symptoms.

CASE REPORT.

S. C. J., male, age 52 years, was brought to the office Oct. 27, 1944. The patient could speak only Spanish, but the county nurse who accompanied him said he claimed that a fly flew into his nose several days previously. Upon examining his nasal passage with a speculum the right side was seen to be filled with worms. These were removed as cleanly as possible with suction, and he was sent home with an anodyne for headache. When he returned the next day the right nasal passage was again filled, and a large ulcerating area could be seen extending up into the right ethmoid cells. No worms could be seen on the left side, but the nasal mucous membrane was edematous. The worms were again removed by suction, and he was referred to the Pima County Hospital on Oct. 28, 1944. The treatment consisted of nasal irrigations of 50 per cent chloroform and water every three hours. Great quantities of worms were removed at each treatment. The particular fly in this case must have been a very prolific one, as several hundred larvae were present. On admission the patient's temperature was 102°, pulse 118, respiration 26, W. B. C. 21,550, 80 per cent neutrophiles, urine and Wassermann were negative. He remained in the hospital 14 days and was finally discharged to return to the office for treatment. During this time the entire septal cartilage and perpendicular plate of the ethmoid were loosened and removed with forceps. The worms then entered the left nasal vestibule, and both middle turbinates and the entire ethmoid cells were destroyed. A large ulcerated area covered the entire nasopharynx, and a deep penetrating wound

extended back into the muscles covering the anterior surface of the cervical vertebrae. After the necrosing process ceased, extreme foul-smelling crust formation, similar to that in ozena, was seen in the nose for months.

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130 South Scott Street.

SPONTANEOUS EXPULSION OF A FOREIGN BODY BY TRANSMIGRATION THROUGH THE NASAL WALL AFTER 28 YEARS.*

GEORGE KELEMEN, M.D.,

Boston, Mass.

Because of the unusual course and the long time necessary for the migration of this foreign body through the nasal wall, this case seems worthy of presentation.

D. D. (R. N. No. 360784), a 34-year-old white laborer, was admitted to the skin clinic (Dr. Francis M. Thurmon) of the Boston Dispensary, Feb. 18, 1944, with a "draining cystic lesion on the nose near the right eye, with rolled borders," of one month's duration. The condition improved under treatment of boric soaks, 5 per cent sulfathiazole ointment and desiccation; on March 31 the depression was covered with a dry crust. The Hinton, Wassermann and Kahn reactions were negative.

The patient was readmitted June 12. At this time the area on the right side of the nose was swollen, discharging, and the surrounding skin swollen and edematous. He was referred to the ear, nose and throat clinic with the diagnosis of infected cyst.

Rhinoscopic examination was essentially normal with the exception of a high deviation to the left. A small preauricular lymph node was palpated on the side of the nasal affection. On the right side of the nasal bridge at the level of the inner canthus, a small elevation was seen which resembled a furuncle. After the crust had been removed, a few drops of thick pus were discharged. The results of an X-ray examination (Dr. Alice Ettinger) were normal and there was "no evidence of osteomyelitis." A sulfafilm dressing was applied. On June 19 a similar and somewhat smaller swelling appeared about 1 cm. below the original location and was also opened by removing a small crust; a few drops of pus were discharged. Again, sulfafilm dressing was applied. When the latter was removed on June 21, both openings appeared united into a furrow about 2 cm. long. On the removed dressing, a long, thin splinter was found, also about 2 cm. long. After 10 days the wound healed completely. When questioned about possible previous nasal injuries, the patient recalled that at the age of six "a rib or tip of an umbrella stuck and broke in his nose."

When he was re-examined 10 months later, April 17, 1945, there was a barely visible scar. The patient had no further complaints since the discharge of the foreign body.

Specimen: The specimen floated on the surface of water. The following pathological report was made (Dr. H. E. MacMahon): "Gross examination: The specimen consists of a small fragment of what appears to

*From the Department of Otolaryngology, Tufts College Medical School and the Massachusetts Eye and Ear Infirmary. Aided by a grant from the Charlton Fund, Tufts College Medical School.

The help of Paul A. Warren, Ph.D., Professor of Botany at Tufts College, in evaluation of the histological sections is gratefully acknowledged.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, April 26, 1945.

be bone or wood. Microscopic diagnosis: Necrotic calcified vegetable matter."

A histological examination of the sections was made (Dr. Paul A. Warren) and the following was reported: "The

Fig. 1. Low power.

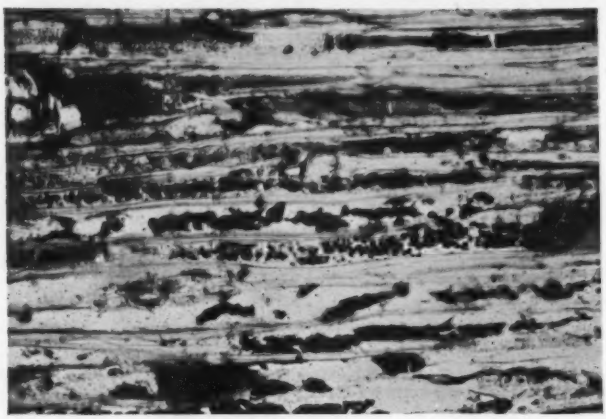

Fig. 2. High power.

sections show the tracheids and wood rays of pine wood in tangential section. Since these cells represent a dead wood tissue, it is not possible that there was any relation between

the cells of the human body and the cells of the wood in the inclusion of the animal tissue found in the tracheids. No explanation can be furnished for the inclusion of animal tissue in these cells beyond the fact that the cells open at the end and could easily have been penetrated by migratory cells."

COMMENT.

In a search for comparable cases in the literature, the following reports were found:

Duration: Erdélyi[5] extracted a piece of a broken adenotome; Kissmann,[8] a vegetable stalk, and Baudet and Lô,[1] a splinter of a shrapnel from the nasal cavity: in these three instances the foreign body had been present for 20 years. Gelfand's[6] patient carried a piece of iron for 23 years, and Krysing's[9] patient, a projectile for 25 years in his nose. Numerous cases have been reported having a duration of less than 20 but more than 10 years. Most foreign bodies, as in this case, caused no symptoms over a long period of time until sudden and acute signs provoked attention and subsequent removal. A duration of 28 years seems to be unequaled.

Transmigration: No report could be found where a foreign body emerged spontaneously after penetrating the whole thickness of the external nasal wall. No scar had been previously noted there, and the wall here is so thin that the splinter could not have lodged between its layers for so long a period. Thus, one has to accept the patient's statement that, at the time of the injury, the piece broke inside his nose and remained there since that time. Other examples of migration are related to a certain distance inside the nose and its surroundings. In a case reported by Stuehmer,[12] a piece of wood penetrated from the left nasolabial fold into the left nasal wall and within two months wandered across the nasal cavity, perforating the septum and reaching the vicinity of the right orbit, when it was finally extracted from between the ethmoidal cells. The case of Combe[3] is an example for movement in the opposite direction: a tin drainage tube has been inserted in an alveolus; lost in the antrum, it took four years before it was spontaneously evacuated through the nasal passages.

Foreign bodies in the nose comprising *parts of an umbrella* have been reported previously on two occasions. Seifert[10]

extracted a piece of wood, part of the cane which had broken inside the nose in the course of a brawl — a counterpart of the case reported in this paper; and Bramson[2] removed a part of an umbrella spring.

CONCLUSIONS.

The vegetable nature of the foreign body would probably have rendered possible its resorption, but as a result of partial calcification it became more resistant. The calcified splinter finally induced irritation with resulting necrosis and destruction of the overlying layers, terminated by the spontaneous expulsion. Though inclusions of a calcareous nature were present, the foreign body did not act as a nucleus for external incrustations by fresh deposits of calcium, producing ultimately a rhinolith (Eggston[4]). There was infiltration but no incrustation by lime salts. It may be mentioned that even rhinoliths, known to have been carried in the nose as long as 60 years (Seifert[11]), never escaped by working their way through the external nasal wall. This is remarkable because instances are known where rhinoliths and concrements of a related nature (Kelemen[7]) penetrated the internal partitions between the nasal and adjacent cavities.

SUMMARY.

A piece of wood from the tip of an umbrella was carried in the right half of the nasal cavity of a 34-year-old white male for 28 years. It became partially calcified and was expelled spontaneously under mild local inflammatory signs with perforation of the nasal wall at the level of the inner canthus. The case is unique both in the length of time the foreign body was carried inside the nasal cavity and in the spontaneous expulsion by transmigration of the entire thickness of the external nasal wall.

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